SINGER AND ROSE-INNES

THE RECOVERY FROM POLIOMYELITIS

A STUDY OF THE CONVALESCENT PHASE

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by

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To

WENDY APTHORP

1943 to 1963

She had infinite courage

FOREWORD

IN the summer of 1956-7 there was an extensive epidemic of poliomyelitis in South Africa. It was particularly severe in Cape Town. To meet the needs of the increasing numbers of paralysed children during this epidemic, the Convalescent Poliomyelitis Unit was established in the Red Cross War Memorial Children's Hospital. This monograph is concerned with a study of the convalescent phase of poliomyelitis and especially with the experience which Mr. Martin Singer and Mr. Peter Rose-Innes gained at this time.

Poliovirus vaccines introduced within the last decade have apparently met an increasingly alarming situation and brought the disease under control. In those countries in which they have been extensively used a notable reduction in the incidence of paralytic cases has been achieved. However, in the countries in which vaccine has not yet been issued, epidemics of increasing severity continue to occur. Even in countries in which the vaccines have been generally available the disease continues to smoulder. This monograph will help those responsible for treatment, especially in early convalescence, to ensure that their patients are being given the best chance of recovery.

J. H. S. GEAR.

1963

PREFACE

THE aim of this monograph has been to collect and collate information, and record some experience of an aspect of poliomyelitis that has been relatively neglected in the vast study of this disease. We have not found a comprehensive text on this subject. The residual deformities of poliomyelitis have received the close attention of orthopaedic surgeons, and the polioviruses and their acute clinical depredations have been studied very extensively. The convalescent period of poliomyelitis, in contrast, has evoked little systematic critical interest.

This should not be so. This period of recovery, in fact, can be considered the most important and rewarding for effective treatment of patients with spinal poliomyelitis. A vigorous, carefully planned orthopaedic regimen during this period has greater value than at any other time. We have attempted to describe the planning, and practice of such a regimen. This was developed in the special unit which was established at the Red Cross Children's War Memorial Hospital, Cape Town, to help cope with an epidemic of poliomyelitis which involved Cape Town and its environs during 1957.

In addition, an introductory historical sketch, which is intended to show some of the many facets of this disease, has been included, and an outline of the pathogenesis. This was felt to be an appropriate background against which to draw an account of the disease dealing with its later stages. This historical and pathological account is an outline only, and hence is in many ways incomplete. It is hoped the reader may be interested in those parts with which he is less familiar, and excuse the omissions in those in which he is expert.

Since the epoch of effective active immunisation against poliomyelitis it is likely that the disease will become a rarity in one or two decades. If this does eventuate this account may have some value as a record. Almost certainly, however, occasional cases will continue to appear, and even epidemics may occur in some countries. This survey may be of some help, then, to those who undertake the care of these patients.

We wish to record our thanks to the Staff of the Engineer's Department who constructed the tank in the pool room and all the special apparatus we requested.

Professor John D. Hansen, Paediatrician, Red Cross Children's War Memorial Hospital, was most helpful and co-operative in handling

all our medical problems and assisting us plan the details of the scheme to combat respiratory infection. We are grateful to Dr. J. Mostert, Superintendent of the Red Cross Children's War Memorial Hospital for his help in the establishment of the unit. Mr. B. Todt, principal clinical photographer at Groote Schuur Hospital, Cape Town, is responsible for all the photographs and we thank him for his skill and patience.

We are also indebted to Professor J. H. Louw, Head of the Division of Surgery, Groote Schuur Hospital, Cape Town, and the University of Cape Town, under whose auspices this unit was established, for enabling us to complete this work; and to Professor C. E. Lewer Allen, Nuffield Professor of Orthopaedic Surgery, University of Cape Town, for his encouragement.

Finally, we would like to thank Mr. W. J. W. Sharrard, Consultant Orthopaedic Surgeon, Royal Infirmary, Sheffield, for his criticisms, and Mr. Charles Macmillan and Mr. James Parker of Messrs. E. & S. Livingstone for their great help and advice.

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Groote Schuur Hospital, and
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CHAPTER 1

HISTORICAL NOTES

THE clinical description of a disease recognised as poliomyelitis began more than one hundred years ago. Since that time both the disease and the knowledge of it have undergone remarkable changes. Few diseases have provoked as immense a study as this and few have proved as intractable to yielding up their secrets. It is only recently that this work at last provided a means of preventing the onslaught of the disease, by the use of an effective vaccine in North America in 1954.

Early Description of the Disease

Two early authors, Underwood (1789) in England and Monteggia (1813) in Italy, commented on a febrile illness in small children followed by paralysis and deformity of limbs. Interest was at first aroused by the deformities of the disease and Shaw in 1823 illustrated a discussion of scoliosis with cases of wasting and paralysis which had occurred in India. During the following twenty years reports appeared more frequently, describing the tendency to contracture and deformities, due to paralysis in infancy, particularly of the lower limbs, for example club foot, which were clearly distinguished from congenital deformities.

In 1840 Heine produced a treatise on paralytic conditions of the lower extremities which laid the foundations for the accurate clinical description of the disease and its differentiation from similar conditions. Then followed the work of Duchenne (1855) and later Charcot (Wickman 1913) in Paris. They advanced knowledge of the basic pathology to a point that was not surpassed before the end of the 19th century.

Duchenne described the disease minutely, emphasising with remarkable insight many of its essential features, such as flaccid paralysis occurring suddenly with fever in infancy; atrophy of the completely paralysed muscles and their infiltration by fat; the concept of deformity due to muscle inbalance; secondary stunting of local skeletal growth; and, most important, destruction of the motor cells of the anterior horns of the spinal cord. He recognised that the localisation and severity of these cord lesions were related to the degree and distribution of the characteristic paralyses. He named the disease Infantile Paralysis and described the same disease in adults as Acute Anterior Spinal Paralysis of the Adult. He used faradisation of muscles extensively in treatment and to test their contractility. Aided

by his electrical studies he developed a crude but fair prognosis for muscle recovery, clearly distinguishing that, although initially many muscles are paralysed, some recover rapidly and completely, others recover partially and more slowly and some remain permanently paralysed.

The quality of his observation is well shown in a comment of his on muscle function '. . . the gravity of the prognosis depends less on the number of muscles affected than on their functional importance . . . the foot is less deformed, and the functions of the lower limb less impeded, by the loss of all the muscles of the foot than by the paralysis of only a few of them . . . when very considerable deformities are gradually produced'.

These concepts of the disease were generally adopted without significant addition during the second half of the century.

It seems that throughout this period the disease occurred sporadically, mainly attacking infants under the age of five years and appearing at random particularly in western Europe and north-eastern America. At first relatively rare, it was frequently encountered by mid-century and accepted as a familiar endemic disease, but was not considered any threat to a community's health (Paul 1955).

However, by the 1860's and 1870's notice had been taken of its increasing incidence, for example by Taylor (1867) in North America.

The Advent of Epidemicity

Although the first reported epidemic occurred on the island of St. Helena in 1834 (Bell 1836), it was not until the 1870's that the disease began to show an alarming epidemic character in Europe. It erupted first in Scandinavia; other parts of western Europe followed in 1880-1890, and north-eastern America in the 1890's. At first these were small limited outbursts, but after 1900 a quickening of tempo seemed to occur. Sporadic infantile paralysis underwent a transition, to become epidemic poliomyelitis throughout Europe and the eastern United States of America, and then beyond, until most of the colder and temperate countries of the northern hemisphere were afflicted by increasingly frequent and increasingly large epidemics.

With the advent of epidemicity the study of poliomyelitis accelerated. At first there was some doubt that, in fact, it was the same disease, but the careful observations of Medin (1890) in a Stockholm epidemic established its identity, that it was undoubtedly infectious, and that, in addition to the well-known spinal form of the disease, bulbar and encephalitic types occurred. The first of the great epidemics which struck Norway and Sweden in 1905 lead to the classic, comprehensive

studies by Wickman (1913). The fundamental importance of his recognition that the disease often occurs in abortive form, and that these cases and healthy contacts are as infectious as frankly paralytic cases, foreshadowed modern epidemiological concepts. He meticulously followed the disease in small isolated communities and showed that its spread radiated from frank cases by close association and movement of person to person, often with a healthy individual or an abortive case as intermediary. As a result of these investigations he advised that cases and contacts should be treated as contagious and isolated; and that intestinal and nasobuccal secretions be carefully destroyed as being highly probable vehicles of the infective agent.

The Change in Age Incidence and Geographic Spread

Since Wickman's time the pattern of epidemic poliomyelitis has evolved further (W.H.O. 1954). It was soon noted that once epidemicity had started in any particular country, the change appeared to be irreversible and the regular recurrence and size of epidemics steadily increased.

In addition a new trend appeared. The disease's incidence, where it had become epidemic, began to spread within the age-span of communities, losing its previous infantile character. Paralytic cases occurred with greater frequency among ever-older age groups in successive epidemics. Today in north-eastern U.S.A. over 35 per cent. of cases occur in those older than 15 years of age, and in Sweden the proportion is even higher (Paul 1955). In many other countries the pre-epidemic infantile characteristics are still predominant.

Geographically there has been a steady extension of epidemicity throughout the world, with one notable irregularity: the change was delayed in most semi-tropical and tropical countries until after 1940. Attempts to relate this to racial immunity and climate failed. Although epidemics occur almost invariably in summer and autumn, they do so everywhere. In addition it became known that poliomyelitis was fully endemic in all these places, but appearing in its mid 19th century form—occasional, sporadic, affecting infants and children under five years—resembling, in fact, classical 'infantile paralysis'.

Since the Second World War epidemics in many of these lands have begun to repeat the evolution shown, say, in western Europe during the previous 50 years (Paul 1955).

A clue to these phenomena was shown in populations made up of communities with widely differing standards of public hygiene. It was found that in these circumstances the stage of evolution reached by poliomyelitis was closely related to the development of each group's

standard of living, particularly their public and domestic sanitation (Sabin 1949; Gear 1955).

The interesting and paradoxical conclusion which emerged was that the more advanced a community's hygiene, the more subject it became to epidemic poliomyelitis, with a higher incidence of paralysis among older individuals, and vice versa.

The Pattern in South Africa

The Republic of South Africa, with its heterogeneous population of contiguous, but separate, colour-race social groups, which have great differences in domestic hygienic environments, and a temperate to sub-tropical climate, has illustrated this evolving pattern clearly (Gear 1948, 1955).

Endemic for many years, poliomyelitis first broke out in epidemic form in 1918. The next attack was in 1944, followed in 1947 and each second or third year thereafter by successive epidemics. The overall incidence of the disease has steadily increased (Fig. 1).

At first the incidence of paralytic cases among Whites was as much as 10 times that among Africans and Coloured who have a far lower standard of living. However during the extensive 1956-7 epidemic the significant change was first noticed that the incidence had become about equal in the two social groups. In Africans and Coloured the disease remains essentially infantile, occurring largely in infants under five years. In Whites, on the other hand, during this epidemic all children's age groups were equally affected, as well as large numbers of adults, in whom the disease tended to be unusually severe.

Advancing Knowledge of the Poliovirus

The first step in isolating the material cause of poliomyelitis was taken by Landsteiner and Popper (1909) when they successfully transferred the disease to rhesus monkeys, by intraperitoneal and intracerebral inoculation of a bacteria-free spinal cord emulsion, obtained from fatal human cases. From this, and similar work by Flexner and Lewis (1909), Romer, Joseph and Levaditi (Wickman 1913), it was inferred that the infecting agent was a filterable virus.

There then followed a period during which advance was hampered by the cost and cumbersomeness of experiments on monkeys, for the virus was found to have a narrow host-range in primates only. It was 30 years before an extra-primate host was found in the cotton rat, a small animal suitable for laboratory use (Armstrong 1939). This gave great impetus to research on the virus and its host response, and later there was further progress after the revolutionary discovery that the

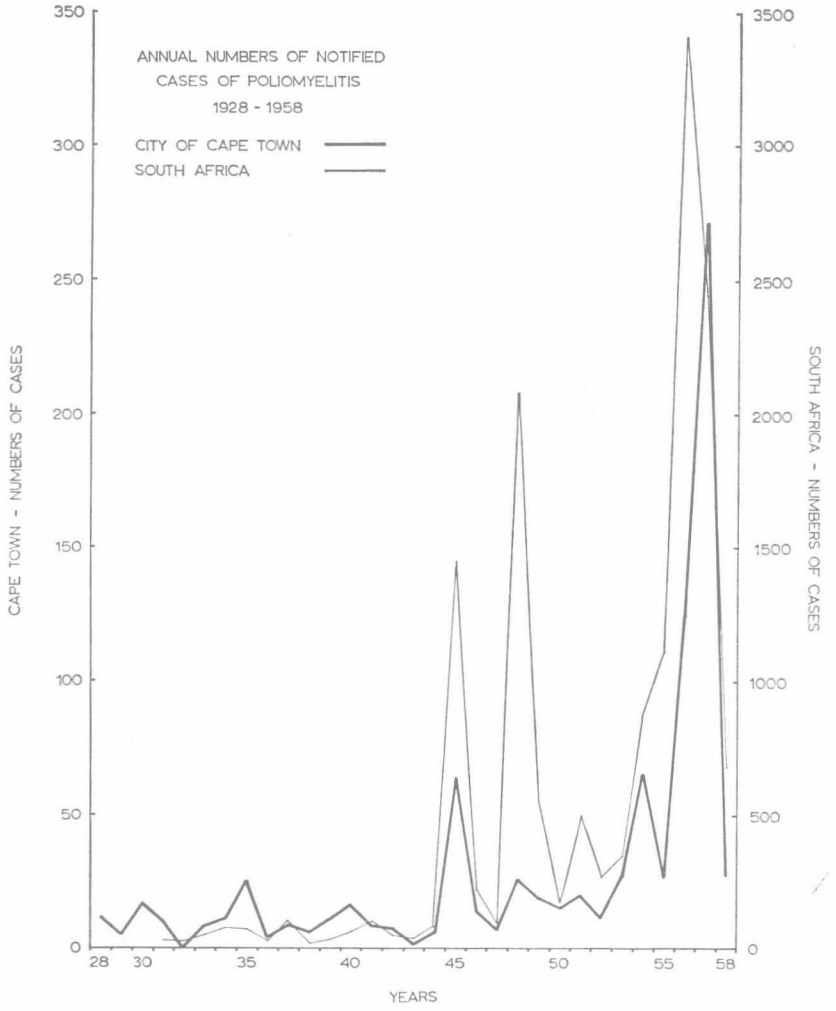

FIG. 1

The annual occurrence of notified cases of poliomyelitis in South Africa, and in the city of Cape Town, during the years 1928 to 1958.

virus could be cultured in vitro in living cells (Enders et al. 1949). Human embryonic tissues were first used, but since then cells from a wide variety of primate organs have been employed (W.H.O. 1954; Enders 1955). Further rodent adaptation of all types of poliovirus has been achieved in mice (Li and Habel 1951; Li and Schaeffer 1953), and recently embryonated eggs have been used to propogate the virus (Roca-Garcia and Jervis 1955).

During much of this time the belief that a single infective agent

was involved confused many of the basic problems of poliomyelitis. In 1931 (Burnet and MacNamara) it was noted that immunologically distinct types of convalescent sera occurred, but not until 1949 (Bodian et al.) was it shown that various strains of the virus could be grouped into three principal immunological types, named Brunhilde, Lansing and Leon, or simply, Types I, II, III, respectively. This was confirmed by an extensive study of more than 100 strains of virus in 1951 (Committee on Typing of the National Foundation).

The analytical definition of the poliovirus has gradually attained a remarkable refinement. High-speed differential centrifugation yielded a relatively pure preparation of virus in 1946 (Loring and Schwerdt). The electron microscope showed the virus as a spherical particle about 25 m/μ in diameter (Sabin et al. 1954; Bachrach and Schwerdt 1954) and it has since been obtained in crystalline form (Schaffer and Schwerdt 1955). It was found to be a nucleoprotein, 20 to 30 per cent. of which consists of ribose nucleic acid. This RNA component appears to be the active pathogenic part of the structure; it alone, separated from the protein component, is capable of infecting susceptible tissues (Colter et al. 1957; Alexander et al. 1958).

The Virus in the Community

An extensive search to discover any natural animal or insect host to the virus failed and it could not be grown in inanimate media. Man became accepted as the only species in which the virus propagates in nature.

However, the central problem of the epidemiology of poliomyelitis —how the virus was transmitted from case to case—remained unanswered for a long time. Its solution required two major revolutions in the concept of the disease's nature.

The easiest method of infecting laboratory monkeys, namely by inoculation into some part of the nervous system, intracerebrally (Flexner and Lewis 1910), or into a large peripheral nerve (Hurst 1930), led to great emphasis being laid on a quality of neurotropism attributed to the virus. It was thought that the virus entered the central nervous system by travelling along nerves and nerve tracts, because of a particular affinity for nervous tissue (Howe and Bodian 1942). This proved in one respect to be a salutary example of the danger of deducing, from animal experiments, the conditions of human disease (W.H.O. 1954). In lower primates, particularly rhesus monkeys, which were used for most experiments, the only atraumatic method of infection that succeeded with some regularity was the intranasal instillation of virus material (Brodie and Elvidge 1934). It was thought,

therefore, that natural infection in man followed a similar route; that the virus entered and left the host only where nerve fibres lay naked to the environment; and that it ascended into the central nervous system via the olfactory nerves.

However, a mass of data accumulated that was contradictory or inappropriate to this thesis. An ever-increasing cognisance was taken of the constant appearance of the virus in pharyngeal secretions and faeces at the time of infection. Epidemiological studies suggested that transmission occurred by intimate hand-to-mouth contagion; that epidemics were related in a curious way to sanitation; that the lower on the primate scale, the more artificial and difficult it was to produce infection, and the less like human poliomyelitis the disease was in many respects.

In 1941 Howe and Bodian produced poliomyelitis in chimpanzees which most closely resembled the human disease in all aspects, by oral feeding of virus, and this paved the way to the concept of poliomyelitis as essentially an alimentary infection, with parenteral invasion of the host from parts of the gastro-intestinal tract (Howe and Bodian 1942; W.H.O. 1954). Evidence that invasion occurred by viraemia now fell more acceptably into place (Bodian 1952; Horstmann 1952; Horstmann and McCollum 1953; W.H.O. 1954).

The second great change in the concept of the disease was due to the recognition that infection was far more widespread in a population than had been thought. The revealing demonstration that poliovirus could be isolated from urban sewage, even in the absence of reported cases (Paul and Trask 1941; Mundel et al. 1946; Gear 1948) suggested an ever-present reservoir of virus in any community. Further, although it had been shrewdly guessed at since Wickman's time, that paralytic cases were only part of the disease's manifestation during epidemics, the true extent of infection was revealed with the advent of tissue culture detection of the virus. It was found that for each paralytic case, there occur from 100 to 1000 non-paralytic or inapparent infections (Howe 1949; Casey et al. 1950). The vast majority of cases of poliomyelitis, in fact, had no clinical disturbance, or less than a common cold caused—but all showed a similar pattern of virus excretion from the alimentary tract, usually lasting three to four weeks (W.H.O. 1954; Paul 1955).

The failure of all attempts to control epidemics by conventional means now became more understandable.

An interesting byway of research was the discovery of a spontaneous encephalomyelitis of mice (Theiler 1937; Theiler and Gard 1940a, 1940b). This virus disease, unlike poliomyelitis in rhesus monkeys,

proved to be an uncannily accurate, diminutive model, in mouse communities, of human poliomyelitis. The study of this disease contributed much to the understanding of these epidemiological aspects of poliomyelitis. Burnet (1945) found this of great general biological interest, and suggested that human poliomyelitis was a disease in evolution, and that the rodent and human viruses have a common ancestral origin.

Immunity

There was early recognition, from the time of the first animal experiments, that one attack of poliomyelitis was commonly followed by immunity to further attacks. Romer and Joseph (1910) showed that serum from convalescent children and monkeys would neutralise the virus in vitro. Subsequently, however, on many occasions, these findings were not confirmed. The ensuing confusion was unexplained for 40 years, until the demonstration that there was no cross-immunity between the three distinct types of virus, although the clinico-pathological effects of each were indistinguishable. It was realised that only the Lansing strain had been used for almost all the experiments. At this time, too, laboratory methods of antibody estimation were greatly facilitated by the use of rodents and tissue culture, and field studies of immunity became possible.

It now emerged that in communities in which poliomyelitis occurred, immunity to all three virus types was acquired by virtually every individual relatively early in life. This confirmed the view that poliomyelitis infection in such communities is universal and that in all but rare instances the attack is silent. It became clear that simple alimentary passage of the virus evoked antibody production. Further, the level of circulating antibody at the time of infection appeared to be one of the decisive factors in determining the severity of that infection's course (W.H.O. 1954).

During the first six to eight months of life infants possess antibodies corresponding to their mothers' types, probably carried across the placenta. Paralytic poliomyelitis is uncommon during this period. The antibody then disappears. Its subsequent reappearance, however, was found to differ significantly in two situations. In communities with primitive sanitation, where poliomyelitis appeared in its classical form—infantile and sporadic—high antibody levels were produced within two or three years. In contrast, this reappearance of immunity was markedly delayed in more highly developed groups, among whom the recurring, epidemic disease was established (Paul et al. 1952; Sabin 1955).

It was now possible to relate many of the diverse features of poliomyelitis' epidemiology (Howe and Wilson 1955). The incidence of paralytic disease in any particular age group was an expression of the overall immunity of that group, and this in turn depended on the opportunities for, or the risk of exposure to the virus. The relative shift in incidence of paralytic disease to older ages in communities with advanced public sanitation, reflected an increasing difficulty in meeting the virus during the early years of life, in a relatively unpolluted environment (Sabin 1949; Gear 1955).

The balance between the immune state of the community and its vulnerability to the virus has been strikingly shown in two instances. In the winter of 1949 a catastrophic outbreak occurred in Canada in a small settlement of Arctic Eskimos, among whom poliomyelitis had been unknown. The attack rate was an unprecedented 25 per cent. in all age groups up to 60 years (Peart 1949). Secondly, during the Second World War a tenfold increase in incidence of paralytic attack was shown among American troops stationed in south-east Asia and the eastern Mediterranean areas, compared with those in continental Europe (Paul 1949).

It has been emphasised recently (W.H.O. 1954; Gear 1955) that, because of the differing opportunities for acquiring natural immunity in various parts of the world today, poliomyelitis has become one of the most serious hazards to health for recently arrived immigrants from Europe and North America into countries where primitive endemic poliomyelitis prevails—The Middle East, North and Southern Africa, and many parts of Asia.

The Treatment of Respiratory Failure

There is little doubt that, throughout the history of poliomyelitis, every established case that has occurred has run a natural course during the acute stage, but for one circumstance. Poliomyelitis seldom causes death, except in that group of cases where central depression of respiration, respiratory muscle paralysis, or bulbar palsy of pharynx and larynx occur. In these the mortality was, until recently, appallingly high, due in part to respiratory insufficiency, but predominantly to a superimposed respiratory obstruction by inhaled vomitus, or the stagnation of secretions (Russell 1956).

Attempts to save these cases began about 1930. At this time Drinker and McKhann (1929) invented the first tank respirator, a device designed to sustain artificial respiration over long periods. The second aspect of the problem was attacked soon afterwards by the introduction of tracheotomy for these cases (Wilson 1932), by

which secretions could be cleared more easily, and the risk of the aspiration of vomitus lessened. The use of these methods was at first slowly adopted, but during the last 15 years they have been vigorously applied, and refined, with gratifying results (Galloway 1943; Galloway and Seifert 1949; Neffson 1952; Russell 1956).

A notable advance during this time has been the development of the method of intermittent positive pressure respiration (IPPR). During the catastrophic 1952 Copenhagen epidemic, Lassen (1953) invited anaesthetists to co-operate in devising a scheme of emergency management for 'respiratory' cases. By means of tracheotomy with a cuffed tube, manual ventilation via the tracheotomy by an anaesthetic bag manned by teams of trained medical students, nurses and laymen, postural drainage and frequent aspiration of the respiratory secretions, the mortality in these cases was reduced from 80 to 25 per cent.

Since then intermittent positive pressure respiration, which combines the advantages of tracheotomy with a simple means of artificial respiration, has evolved, particularly in the use of mechanical pumps to provide prolonged ventilation; and the indications for its use, as well as the use of tank respirators, have been clarified, with great saving of life (Bang 1953; Russell and Schuster 1953; Crampton-Smith et al. 1954; Spalding and Young 1955; Lassen 1955; Russell 1956).

Other Methods of Treatment and Control

No specific method of treatment has been found of any avail in altering the essential course of poliomyelitis once infection has occurred.

There have been enthusiasms for a variety of treatments for the acute phase; examples range from blistering the back and parenteral strychnine, at the end of the 19th century, to, more recently, repeated or continuous drainage of spinal fluid, intravenous administration of hypertonic solutions, and intrathecal adrenalin. Additionally antibiotics were found ineffective. During the 1930's and 1940's there was a great vogue for Sister Kenny's idea of treatment by the application of hot packs, said to be based on a new concept of the disease which denied that true paralysis occurred; muscle spasm, inco-ordination and mental alienation were the real disabilities, it was believed. None of these methods has proved of value, and they have fallen into disuse (Howe and Wilson 1959).

Tribute should be paid, however, to the simple symptomatic relief of pain, discomfort and fear, where this has been given to poliomyelitis victims, by good nursing and common-sense doctoring over the years.

The use of convalescent serum, and gamma globulins extracted from pooled adult serum, disappointingly, was shown to have no